LEAD EXPOSURE INVESTIGATION AND FOLLOW-UP PROTOCOL FOR NUNAVIK

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FOREWORD

Due to extensive use of the two units in the scientific literature, lead concentrations are measured using the metric system (µmol/L) with conversion to µg/dL. Remember that 0.48 µmol/L equals 10.0 µg/dL, and that for an approximate conversion from µmol/L to µg/dL, multiply by 20.

INTRODUCTION

The effects of exposure to lead on human health have been known for a long time and were first demonstrated among populations of workers. Then, in the early 1980's, public health authorities became concerned with environmental contamination in terms of neurological effects on the foetus and on young children (Needleman and Bellinger 1991; Davis et al. 1993; Goyer 1993).

Data from two recent studies in Nunavik, one among the adult population (Health Québec 1994) and the other on samples of umbilical-cord blood (Dewailly et al. 1998) reveal a problem of exposure to lead among the population. Moreover, samples taken in 1997-98 in certain villages on the Hudson coast, at the request of referring physicians on the basis of clinical suspicions, showed alarming levels of blood lead in several subjects.

This document aims at providing guidelines to assist Nunavik clinicians in interpreting the results of blood lead analysis and managing the patients whose concentrations require follow-up.

1. TOXICO-KINETICS AND HEALTH EFFECTS

With the permission of Dr. Robert Plante, coordinator of the work group that produced the document, *Critère d'une intoxication et d'une exposition significative : le plomb* [Criteria for lead poisoning and significant exposure] (Plante et al. 1998), as a tool for determining declaration criteria for the reportable diseases file in Québec, we have reproduced, almost in full within this section, Parts 1.2, 1.3 and 1.4 of said document; here they become Subsections 1.1, 1.2 and 1.3.

1.1 Toxico-Kinetics of Lead in the Human System

Several properties of lead's toxico-kinetics and its inorganic derivatives vary according to the subject's age. While the proportion of ingested lead absorbed in an adult system is normally only 8% or 9% (4% to 11%) (Chamberlain 1978;

Rabinowitz et al. 1980; Watson 1986), it can reach 42% to 66% in a child (Alexander et al. 1974; Chamberlain 1978; Ziegler 1978). Intestinal absorption of lead is affected by the type of derivative ingested and the gastrointestinal contents; is is also facilitated by certain nutritional deficiencies (involving iron and calcium in particular). The proportion of ingested lead absorbed diminishes slowly up to the age of two years and then more rapidly after that, reaching the adult absorption level at the age of about 10 years (O'Flaherty 1995). Poisoning through ingestion is therefore much more frequent among young children than among adults, and all the more because young children tend to carry objects around in their mouth.

The proportion of inhaled lead absorbed by the lungs is estimated at 50% (Rabinowitz et al. 1977) but varies according to the size of the particles inhaled; almost all the lead deposited in the alveoli is absorbed. Part of the absorbed dose is distributed in tissue such as the liver, kidneys and other soft tissues; the majority is deposited in the bones while part is eliminated in urine and feces.

Lead is metabolized in the same way as calcium during mineralization of bone tissue and remains there until the bone is resorbed, and this is how it accumulates. It is estimated that the bones contain 95% of the total body lead content in adults and 75% in children. As the phenomenon of resorption slows down considerably when growth stops, the half-life of lead stored in bone tissue is estimated at 20 to 25 years in adults and about one year in children (O'Flaherty 1995). Some months after the end of chronic exposure, the lead concentration in bone tissue attains equilibrium with that in the blood; when exposure stops, a child's blood lead content therefore drops much more rapidly than that of an adult.

Lead is eliminated in a triphasic process: the first phase, a rapid one, involves blood lead and lead deposited in certain soft tissues (half-life of about 35 days); the second phase, a slower one, involves lead stored in trabecular osseous tissue; the third phase, a very slow one, eliminates lead stored in cortical osseous tissue (half-life of 20 to 25 years in adults). Duration of half-life is particularly affected by the total bodily content, which depends on the duration and intensity of exposure.

Even though exposure to lead decreases as pregnancy develops, the level of blood lead content increases, especially from the 20th week onward, when it reenters the circulation from bone deposits. The lead crosses the placental barrier to reach the foetus, whose blood lead content approaches that of the mother (Lagerkvist 1996; Goyer 1996). Furthermore, breast milk is contaminated in proportion to the mother's bodily concentration. During pregnancy and breast-feeding, any exposure for the mother thus directly

affects the infant, which can represent a significant part of the lead measured in the latter.

1.2 Effects on Health

The symptoms of acute poisoning, which arise after short-term exposure to very high environmental concentrations, are almost never seen anymore in economically developed countries where health measures are in effect; however, they still occur episodically among certain workers exposed to very high concentrations of lead. This type of poisoning is characterized especially by epigastric and abdominal pain, vomiting, renal damage and sometimes hepatic damage; convulsions and coma leading to death have been described. On the other hand, chronic poisoning most often results from prolonged occupational exposure to ambient lead concentrations at levels sufficient to maintain blood lead content at higher than 1.5 µmol/L (31.3 µg/dL) over several years, indeed, decades. While it often occurs unnoticed, this chronic poisoning, however, can lead to general health problems (headaches, loss of appetite, mood swings, lowered psychomotor performance, colic or abdominal pain, weight loss, pallor, weakness, frequent muscular rheumatism), damage to the hematopoietic system (mild normocytic or hypochromic anemia) and damage to the reproductive system (hypospermia, increased risk of miscarriage) (Lauwerys 1990). Chronic poisoning has been associated with high blood pressure as well as damage to the renal system and peripheral nervous system (reduced conduction speed of the nerve impulse in the peripheral motor nerves).

Among young children, acute poisoning is still occasionally seen with the clinical symptoms described above and generally occurs at blood lead concentrations higher than 1.5 µmol/L (31.3 µg/dL); anemia and encephalopathy are particularly present. Research during the past decade has shown that after prolonged exposure to lead, children whose blood lead content is higher than 0.48 µmol/L (10.0 µg/dL) are more likely to present learning difficulties and behavioural problems (Centres for Disease Control 1997). That its effects appear insidiously without necessarily being detected clinically does not reduce the problem's gravity. To date, we are unable to define a precise level of blood lead content having no health effects, especially on the development of young children. All recent knowledge therefore dictates prudence, and the objective of public health authorities is to reduce, as much as possible, the level of exposure to lead for all children, without omitting exposure during the particularly critical period of foetal life. As children grow, several changes reduce their vulnerability: they carry objects less often in their mouths, the absorption rate of ingested lead drops and their nervous system matures.

1.3 Relationship between Level of Blood Lead and Health Effects

Several studies have demonstrated that there is a solid correlation between blood lead content and health effects; in certain cases, however, impregnation of the organism may last a long time before symptoms become apparent. Table 1 presents the relationship between blood lead and health effects, in both children and adults.

TABLE 1: EFFECTS OF INORGANIC LEAD ON CHILDREN AND ADULTS (MINIMUM CONCENTRATION PRODUCING AN OBSERVED HARMFUL EFFECT[1])

Child	Blood Lead Content µmol/L (µg/L)	Adult
	7.0 (1 400)	
	5.0 (1 000)	⇐Encephalopathy
Encephalopathy/nephropathy/obvious anemia⇒		
		⇐Obvious anemia
Colic⇒		
	2.5 (500)	⇐↓Hemoglobin synthesis
↓Hemoglobin synthesis⇒	2.0 (400)	⇐Peripheral neuropathy/nephropathy
Metabolism of vitamin D[2] (change)⇒		⇐Effects on reproduction
	1.5 (300)	
		⇐↑Erythrocytic protoporphyrin (men)
↓Nerve conduction velocity⇒		
	1.0 (200)	
↑Erythrocytic protoporphyrin⇒		
	0.75 (150)	
		⇐↑Erythrocytic protoporphyrin (women)
Metabolism of vitamin D[2] (change)⇒		
Toxicity related to development⇒	0.5 (100)	
↓I.Q.[2]		
		⇐Hypertension[2]

Source: Lévesque, B., et al. *Protocole d'investigation et de suivi en regard de l'exposition au plomb au Nunavik*. Québec: Direction de santé publique de Québec, March 1999.

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[1] Reproduction of the ATSDR adaptation, *Toxicological Profile for Lead (1990)*, by the Federal-Provincial Committee on Environmental and Occupational Health, September 1994.

[2] No minimum value has been discovered yet.

2. NORMS AND REFERENCE LIMITS

On the basis of lead's harmful effects on a developing nervous system, Health Canada has established that the populations at risk are women of child-bearing age and children living in communities likely to be exposed to lead (Health Canada 1994). Thus, in 1991, the Centres for Disease Control (CDC) decreed that for children and foetuses, blood lead concentrations as low as 0.48 µmol/L (10.0 µg/dL) are associated with harmful effects, and that even though the effects of low exposure to lead may seem negligible for one individual, they can have a significant impact on a community. Preventive activities in programs dealing with lead exposure should also aim at reducing blood lead concentrations in children to below 0.48 µmol/L (10.0 µg/dL) (CDC 1991). In Canada, it is estimated that if the average blood lead content in children exceeds the general population's average by more than three times the standard deviation, or if the percentage of children whose blood lead content is higher than 0.48 µmol/L (10.0 µg/dL) reaches double that of the general population, it will be necessary to apply a community program to identify and reduce the sources of exposure (Health Canada 1994).

Among adults, the principal concern is for workers affected by situations exposing them to lead. Aside from exposure of pregnant women, who are protected in Québec by the *Act respecting occupational health and safety* (Québec 1997), which covers the preventive withdrawal of the pregnant or breast-feeding worker, norms for blood lead content are used to protect workers. In Québec, the *Regulation respecting the quality of the work environment* (Québec 1995) provides for withdrawal from exposure at 1.92 µmol/L (40.0 µg/dL) and return to the initial position at 1.44 µmol/L (30.0 µg/dL). Moreover, the American Conference of Governmental Industrial Hygienists (ACGIH) has set a maximum value of 1.44 µmol/L (30.0 µg/dL) (ACGIH 1998), while the Occupational Safety and Health Administration (OSHA) has decreed that a concentration of 2.88 µmol/L (60.0 µg/dL) requires immediate withdrawal and a value of 1.92 µmol/L (40.0 µg/dL) allows reintegration (OSHA 1999).

3. EXPOSURE TO LEAD IN NUNAVIK

As previously mentioned, evidence for significant exposure to lead for the Nunavik population primarily comes from two sources: data from the *Health Québec Survey* carried out in the early 1990's among the adult population (Health Québec 1994) and the study on umbilical-cord blood samples gathered throughout the Nunavik territory from 1993 to 1996 (Dewailly et al. 1998).

3.1 Health Québec

For the *Health Québec Survey among the Nunavik Inuit* (Health Québec 1994), 305 households in total were chosen at random from among 1 378 in the region's 14 villages. Individuals aged 18 years or over were asked to respond to a series of questionnaires on their health and eating habits. The participants also underwent a physical examination and were asked to go through with a venipuncture for a blood sample. Lead concentrations were thus determined for 492 persons, or 64.4% of the eligible participants.

For all the samples, the arithmetic mean was 0.49 µmol/L (10.1 µg/dL), and the concentrations varied from 0.04 µmol/L (0.83 µg/dL) to 2.28 µmol/L (47.5 µg/dL). Average levels were higher among men than among women--0.52 µmol/L (10.8 µg/dL) compared to 0.45 µmol/L (9.4 µg/dL)--and increased proportionately with age: 0.36 µmol/L (7.5 µg/dL) for those aged 18 to 24, 0.49 µmol/L (10.2 µg/dL) for those aged 25 to 44 years and 0.63 µmol/L (13.1 µg/dL) for those aged 45 to 74.

Among women aged 18 to 44 years, 26% had concentrations equal to or higher than 0.48 µmol/L (10.0 µg/dL). Finally, average blood lead content was higher among inhabitants of the Hudson coast, at 0.55 µmol/L (11.4 µg/dL), compared to 0.40 µmol/L (8.3 µg/dL) for residents of the Ungava coast.

To our knowledge, in Canada, only one population study has been performed on a sampling of the general adult population without an obvious source of exposure. This research was carried out in Alberta in 1980, with a total of 338 samples gathered. The average blood lead concentration was 0.49 µmol/L (10.1 µg/dL), and the values went from 0.05 µmol/L (1.0 µg/dL) to 1.20 µmol/L (25.0 µg/dL) (Health Canada 1994). In the United States, a study by the National Health and Nutrition Examination Surveys (NHANES) conducted between 1988 and 1991 on a random sampling of more than 13 000 persons, of whom more than 9 000 were 12 years old or over, revealed geometric mean concentrations of 0.08 µmol/L (1.6 µg/dL), 0.13 µmol/L (2.6 µg/dL) and 0.19 µmol/L (4.0 µg/dL) respectively for the groups aged 12 to 19, 20 to 49 and 50 years and over. Only 0.5% of women aged 12 to 49 had a blood lead concentration of 0.48 µmol/L (10.0 µg/dL), and this prevalence varied little according to race and ethic group (Brody et al. 1994).

Blood lead values documented among Nunavik residents at the beginning of the 1990's are therefore similar to those of the Alberta population in the early

1980's but higher than the American data, which are more comparable strictly at the temporal level.

3.2 Umbilical-Cord Blood

The results presented in this section come from the report entitled, *Évaluation de l'exposition prénatale aux organochlorés et aux métaux lourds chez les nouveau-nés du Nunavik, 1993-1996* [Assessment of prenatal exposure to organochlorines and heavy metals among Nunavik newborns] (Dewailly et al. 1998). This study was conducted between November 1993 and December 1996 among the resident populations of the 14 Nunavik communities. One of the objectives of that study was to evaluate lead concentrations in the umbilical-cord blood among Inuit newborns in Nunavik.

In total, 491 women participated in the study, 52.1% from the Hudson subregion and 47.9% from the Ungava subregion. Table 2, taken from the report (Dewailly et al. 1998), presents the distribution of newborns according to subregion and village of residence. The average age of the mothers was 24 years, varying from 15 to 42. Out of the total, 85.5% of the mothers who agreed to respond to the question on smoking (n = 373) affirmed having smoked during pregnancy.

Overall, 475 newborns (96.7%) were evaluated for exposure to lead. The geometric mean for cord lead content was 0.19 µmol/L (4.0 µg/dL) [confidence interval 95% (CI 95%): 0.18 µmol/L (3.8 µg/dL) to 0.20 µmol/L (4.2 µg/dL)], varying from 0.04 µmol/L (0.8 µg/dL) to 1.31 µmol/L (27.3 µg/dL). The geometric mean was slightly higher in the Hudson subregion [0.20 µmol/L (4.2 µg/dL), CI 95%: 0.18 µmol/L (3.8 µg/dL) to 0.22 µmol/L (4.6 µg/dL)] than in the Ungava subregion [0.18 µmol/L (3.8 µg/dL), CI 95%: 0.16 µmol/L (3.3 µg/dL) to 0.19 µmol/L (4.0 µg/dL)]. Blood concentrations equal to or higher than 0.48 µmol/L (10.0 µg/dL) were observed among 6.9% (n = 33) of Inuit newborns in Nunavik. The authors noted that the blood lead content among Inuit newborns, on average, was higher in June and July. The average levels measured during those months varied between 0.23 µmol/L (4.8 µg/dL) and 0.24 µmol/L (5.0 µg/dL); during the other months, the lead levels in cord blood varied from 0.17 µmol/L (3.5 µg/dL) to 0.21 µmol/L (4.4 µg/dL).

Blood lead concentrations among Inuit newborns were approximately twice as high as those reported for newborn populations in southern Québec (Rhainds et al. 1999). Likewise, the proportion of newborns with a blood lead content equal to or higher than 0.48 µmol/L (10.0 µg/dL) was 7% in Nunavik

compared to less than 1% for the rest of the province. However, comparisons of umbilical-cord blood lead levels between Nunavik and other industrialized regions in the world are less spectacular. The table in Appendix 1 (Rhainds and Levallois 1993) shows that the levels of cord blood lead in Nunavik are half as high as those reported over the past 30 years, especially before the 1980's. In addition, two recent studies carried out in 1990 in Lucknow (India) (Saxena et al. 1994) and 1993 in Shanghai (China) (Xiao-Ming et al. 1997) report levels of lead contamination among newborns which are clearly higher than those observed in Nunavik. The average cord blood concentrations (geometric mean) in Lucknow and Shanghai, respectively, were 0.80 µmol/L (16.7 µg/dL) (n = 189) and 0.50 µmol/L (10.4 µg/dL) (n = 348). In Lucknow, 70% of the newborns exceeded the threshold value of 0.48 µmol/L (10.0 µg/dL), while in Shanghai, this proportion was 41%.

TABLE 2: NUMBER OF NEWBORNS, BY SUBREGION AND MOTHER'S VILLAGE OF RESIDENCE[3]

Subregion and Village of Residence	n	%
Ungava	**225**	**50.7**
Kuujjuaq	92	20.7
Tasiujaq	13	2.9
Kangirsuk	27	6.9
Kangiqsujuaq	23	5.2
Kangiqsualujjuaq	40	9.0
Aupaluk	9	2.0
Quaqtaq	21	4.7
Hudson	**219**	**49.3**
Kuujjuaraapik	16	3.6
Umiujaq	12	2.7
Inukjuak	49	11.0
Puvirnituq	73	16.4
Akulivik	22	5.0
Ivujivik	16	3.6
Salluit	31	7.0

[3] Dewailly et al. 1998.

3.3 Other Data

Blood samples were taken to measure lead concentrations among residents of certain villages on the Hudson coast, based on clinical suspicions by the referring physicians. In total, close to 200 persons (adults and children) were tested; 15 had levels equal to or higher than 1.0 µmol/L (20.8 µg/dL), while four had levels higher than 2.0 µmol/L (41.6 µg/dL).

4. SOURCES OF LEAD IN NUNAVIK

In general, the air we breathe, the water we drink, the food we eat and the soil represent the principal sources of lead for humans (Health Canada 1994). In the past, due to lead additives in gasoline, air was a major source of exposure for the population; this was clearly demonstrated by the reduction in blood lead levels after the progressive elimination of leaded gasoline, up to its total ban in 1990. It is also quite unlikely for this source to be significant in the current context in Nunavik.

Smoking, whether passive or active, may contribute to lead intake. Several studies have established a dose-effect relationship between the number of cigarettes smoked during pregnancy and lead concentrations in the umbilical cord (Grandjean et al. 1992; Rhainds and Levallois 1992; Rhainds and Levallois 1997). The same relationship was also demonstrated between smoking at home and the blood lead levels in both preschool and school-aged children (Willers et al. 1988). However, the percentage change explained by smoking is relatively low (< 6%). The 1992 Health Québec study revealed that 70% of the Inuit population aged between 15 and 40 years smokes a daily average of 15 cigarettes and that 80% of young women (15 to 24 years) in Nunavik smoke (Health Québec 1994). However, the same study revealed average blood lead concentrations of 0.48 µmol/L (10.0 µg/dL) among smokers and 0.38 µmol/L (8.0 µg/dL) among non-smokers, a slight difference that cannot really explain the scope of the problem of lead poisoning among humans in Nunavik.

In general, drinking water represents a minimal source of exposure to lead for Canadians (Health Canada 1994). When the problem arises, it principally involves old distribution systems constructed or welded with lead (now practically prohibited) (Health Canada 1994). In Nunavik, drinking water is drawn from lakes and rivers, transported by trucks and pumped into plastic reservoirs in the residents' homes. Thus, from the community point of view, this source of contamination seems very unlikely.

The principal source of exposure to lead in the United States is the ingestion, by young children, of paint dust and fragments containing lead. The addition of lead to paint has been banned in Canada since 1972 (Feldman and Randel 1994) and, with a few exceptions, the houses in Nunavik were built after that date.

The contamination of soil, and subsequently the dust in houses, is generally the result of industrial sources in close proximity, such as foundries and battery-recycling plants. These types of industry do not exist in Nunavik.

Food is now the most serious hypothesis to consider. With the reduction in the use of lead solder in sealing food cans--the principal source of exposure involving food for Canadians--the ingestion of lead through consumption of food has dropped considerably over the past years (Health Canada 1994). However, the consumption of traditional foods distinguishes the Inuit from other Canadians. A study carried out among the Qikiqtarjuak population on Baffin Island concluded that the children, women and men of this community ingest lead in their food, with average daily quantities of, respectively, 21.0, 44.0 and 54.0 µg, which translate into weekly doses of 4.6, 4.4 and 4.4 µg/kg, well within the tolerable dose of 25 µg/kg per week established by the World Health Organization (WHO) (Chan et al. 1995). However, this population does not eat fowl. The Inuit of Nunavik, especially those on the Hudson coast, consume important quantities of meat from geese, ducks and ptarmigan. With the data on consumption from the *Health Québec Survey* (Health Québec 1994) and the concentrations measured in the meat of various types of fowl consumed by the Nunavik population, the lead intake for women between 18 and 39 years was determined as originating primarily from the consumption of Canada geese (1.47 µg/day) and ptarmigan (1.07 µg/day). Compared to the Canadian average of 125 µg/day (Chan et al. 1995), this is a relatively minor quantity. Obviously, there are problems with anemia and low calcium intake in Nunavik (Hodgins 1997). These two factors are associated with increased absorption of lead in humans (Mahaffey 1995). Nevertheless, if we examine all the data from a global point of view, the intake of lead contained in traditional foods (contamination of wildlife and quantities ingested) do not suffice in explaining the levels of lead blood among the Nunavik Inuit.

Lead pellets have regularly been discovered by chance in the abdominal radiographs of Nunavik residents. This problem has also been identified among the Cree of northern Ontario (Tsuji and Nieboer 1997). Several studies have revealed that the ingestion of lead pellets or other lead objects can lead to increased blood lead content. In Denmark, it was demonstrated that merely two lead pellets trapped in the appendix suffice to increase the blood level concentration to 0.5 µmol/L (10.0 µg/dL) (Madsen et al. 1988).

Thus, it is very plausible that the ingestion of lead shot (principally used in hunting game birds) or meat contaminated with lead shot during the kill is behind the blood lead content identified among the Nunavik population. To that effect, a recent study determined the radio isotopes of lead (to identify the metal's source) in 60 umbilical-cord blood samples from deliveries performed in Nunavik. The average 206/207 ratio was 1.195, almost identical to 1.193, which was measured in ammunition from four brands used by Nunavik hunters (Lévesque et al. 1998). Thus, it is very plausible that lead shot from ammunition used in game bird hunting is, from a collective point of view, a major source of human exposure to lead in Nunavik. Obviously, except at the individual level, other sources, such as children's ingestion of lead sinkers used for fishing (Mowad et al. 1998) or handling of lead articles in various hobbies, have not been excluded as being potentially significant.

5. CLINICAL GUIDE FOR ASSESSING BLOOD LEAD CONTENT IN NUNAVIK

The present guide was developed for physicians clinically investigating cases of elevated blood lead content in Nunavik. The various elements to consider for case management are presented in Table 3. The environmental assessment in the table refers to the use of a questionnaire to identify the source or sources of lead in the patient's environment (see Appendix 2). This tool (to be developed) will subsequently be used to offer counselling to persons with an elevated blood lead content to reduce their exposure to this contaminant. The systematic screening for blood lead content among pregnant women in Nunavik was not considered in the present clinical guide for several reasons. First, the studies carried out in Nunavik (Health Québec 1994; Dewailly et al. 1998) show that blood lead concentrations among women and newborns rarely exceed 1.0 µmol/L (20.8 µg/dL). In this context, the probability of finding a case of elevated blood lead content is very low. Second, with the existing knowledge, it is not possible to offer adequate, post-screening counselling. What should be done for a case of blood lead concentration higher than 1.0 µmol/L (20.8 µg/dL)? For example, there is no reason whatsoever to recommend abortion even though certain studies have associated a reduction in birth weight and increased chances for a premature birth with exposure to lead. Third, the cost-benefit ratio associated with such screening, a priori, does not appear to be very effective. We believe that in the case of pregnant women, primary prevention, including the search for the sources of lead exposure and concrete actions on those sources, is a much better choice than screening.

Blood Lead Higher than 2.0 µmol/L (41.7 µg/dL)

- As a priority, search for the source or sources of lead in the patient's environment, using the questionnaire.
- Ensure counselling according to the source or sources identified, with the goal of modifying the environment or lifestyle.
- Repeat the blood lead test as soon as possible.
- Follow the steps below in the case of blood lead content greater than 2.0 µmol/L in two different blood samples.
- Proceed with medical evaluation with the following examinations:
    - complete blood count (investigate for anemia);
    - serum iron;
    - ferritin;
    - % saturation of serum iron;
    - FEP (free erythrocite protoporphyrin);
    - ZPP (zinc protoporphyrin);
    - abdominal radiograph (search for presence of lead shot in digestive tract).
- Refer to toxicologist for assessment of chelation indications.
- Follow recommendations of toxicologist in case of chelation.
- Perform control test for blood lead according to recommendations of toxicologist.
- Testing recommended for blood lead among other family members.

Blood Lead between 1.0 µmol/L (20.8 µg/dL) and 2.0 µmol/L (41.7 µg/dL)

- As a priority, search for the source or sources of lead in the patient's environment, using the questionnaire.
- Ensure counselling according to the source or sources identified, with the goal of modifying the environment or lifestyle.
- Proceed with medical evaluation with the following examinations:
    - complete blood count (investigate for anemia);
    - serum iron;
    - ferritin;
    - % saturation of serum iron;
    - FEP;
    - ZPP;
    - abdominal radiograph (search for presence of lead shot in digestive tract).

- If ferritin < 10.0 µg/L:
    - Administer iron for three months and, subsequently, control blood lead content, blood count, FEP and ZPP.
- If ferritin normal with FEP > 35.0 mg/dL and/or ZPP > 2.5 µg/g:
    - Refer to toxicologist for assessment of chelation indications.
    - Perform control test for blood lead according to toxicologist's recommendations.
- If ferritin normal:
    - Perform control test for blood lead every three months for pregnant women and children aged five or under.
    - Perform control test for blood lead in six months for all other cases.
- Testing recommended for blood lead among other family members.

Blood Lead between 0.48 µmol/L (10.0 µg/dL) and 0.9 µmol/L (18.8 µg/dL)

- Search for the source or sources of lead in the patient's environment, using the questionnaire.
- Ensure counselling according to the source or sources identified, with the goal of modifying the environment or lifestyle.
- No control test for blood lead.
- Optional testing for blood lead among other family members according to environmental assessment.

TABLE 3: ELEMENTS TO CONSIDER IN ASSESSING BLOOD LEAD (PB) CONCENTRATION

Blood Lead Content (μmol/L)	Repeat Blood Lead Test	Environmental Assessment (Sources of Pb)	Other Examinations	Blood Lead Test among Other Family Members	Control Test for Blood Lead	Toxicology Assessment
< 0.5	no	no	no	no	no	no
0.5 - 0.9	no	yes	no	no	no	no
1.0 - 2.0	no	yes	Investigate anemia: • complete blood count • serum iron • ferritin • % saturation • FEP • ZPP Abdominal radiograph (Pb in appendix)	yes	Three months: • ≤ 5 years • pregnant women Six months: • others	no
> 2.0	yes	yes	Investigate anemia: • complete blood count • serum iron • ferritin • % saturation • FEP • ZPP Abdominal radiograph (Pb in appendix)	yes	Follow toxicologist's recommendations	yes

BIBLIOGRAPHY

Alexander, F.W., et al. 1974. Mineral and trace-metal balances in children receiving normal and synthetic diets. Q J Med. 169: 89-111.

Brody, D.J., et al. 1994. Blood lead levels in the U.S. population. JAMA. 272: 277-283.

Centers for Disease Control and Prevention (CDC). 1997. Update: blood lead levels--United States, 1991-1994. Mor Mortal Wkly Rep. 46: 141-146.

Chamberlain, A.C. *Investigations into lead from motor exhausts*. AERE Report R9198. London: HMSO, 1978.

Chan, H.M., et al. 1995. Assessment of dietary exposure to trace metals in Baffin Inuit food. Environ Health Perspect. 103: 740-746.

Comité médical provincial en santé au travail du Québec (CMST). *Guide de pratique professionnelle ; la prévention et le contrôle des intoxications par le plomb en milieu de travail : seuils de déclaration, d'intervention et seuil de retrait préventif du travailleur ou de la travailleuse exposés à un contaminant* [Professional's guide to the prevention and control of lead poisoning in the workplace: levels for declaration, intervention and preventive withdrawal of the worker exposed to a contaminant]. Québec, 1996.

Davis, J.M., et al. 1993. Current issues in human lead exposure and regulation of lead. Neurotoxicology. 14: 15-28.

Dewailly, E., et al. *Évaluation de l'exposition prénatale aux organochlorés et aux métaux lourds chez les nouveau-nés du Nunavik, 1993-1996* [Assessment of prenatal exposure to organochlorines and heavy metals among Nunavik newborns, 1993-1996]. Québec: Centre de santé publique de Québec, 1998.

Feldman, W., and P. Randel. "Screening children for lead exposure in Canada." In *Canadian Guide to Preventive Clinical Health Care*. Ottawa: MAS, 1994.

Goyer, R.A. 1993. Lead toxicity: current concerns. Environ Health Perspect. 100: 177-187.

---. 1996. Results of lead research: Prenatal exposure and neurological consequences. Environ Health Perspect. 104 (10): 1050-1054.

Grandjean, P., et al. 1992. Impact of maternal seafood diet on fetal exposure to mercury, selenium, and lead. Arch Environ Health. 47 (3): 185.

Health Canada. Federal-Provincial Committee on Environmental and Occupational Health. *Blood lead intervention levels and strategies: update of evidence for low-level effects of lead and blood lead intervention levels and strategies*. Ottawa, 1994.

Health Québec. Ministère de la Santé et des Services sociaux. *A Health Profile of the Inuit: Report of the Santé Québec Health Survey among the Inuit of Nunavik, 1992*. Montréal, 1994.

Hodgins, S. *Health and what affects it in Nunavik: How is the situation changing?* Kuujjuaq: Department of Public Health, Nunavik Regional Board of Health and Social Services, 1997.

Lagerkvist, B.J., et al. 1996. Increased blood lead and decreased calcium levels during pregnancy: a prospective study of Swedish women living near a smelter. Am J Public Health. 86 (9): 1247-52.

Lead poisoning among Inuit children: Identification of sources of exposure. Papers from the 19th Annual Meeting of The Society of Environmental Toxicology and Chemistry. Charlotte, 1998.

Madsen, H.T., et al. 1988. Blood lead levels in patients with lead shot retained in the appendix. Acta Radiol. 29: 745-746.

Mahaffey, K.R. 1995. Nutrition and lead: strategies for public health. Environ Health Perspect. 103 (6): 191-196.

Mowad, E., et al. 1998. Management of lead poisoning from ingested fishing sinkers. Arch Pediatr Adolesc Med. 152: 485-488.

Needleman, H.L., and D. Bellinger. 1991. The health effects of low level exposure to lead. Annu Rev Public Health. 12: 111-140.

O'Flaherty, E.J. 1995. Physiologically based models for bone-seeking elements. V. Lead absorption and disposition in childhood. Toxicol Appl Pharmacol. 131: 297-308.

Québec. Ministère de la Santé et des Services sociaux. *An Act respecting occupational health and safety*. R.S.Q. , c. S-2.1. Québec, 1981.

Québec. Ministère de la Santé et des Services sociaux. *Critères d'une intoxication et d'une exposition significative : le plomb* [Criteria for lead poisoning and significant exposure to lead]. Québec, 1998.

Rabinowitz, M.B., et al. 1980. Effect of food intake and fasting on gastrointestinal lead absorption in humans. Am J Clin Nutr. 33: 1784-1788.

Rabinowitz, M.B., et al. 1977. Magnitude of lead intake from respiration by normal man. J Lab Clin Med. 90: 238-248.

Rhainds, M., and P. Levallois. 1997. Effects of maternal cigarette smoking and alcohol consumption on blood lead levels of newborns. Am J Epidemiol. 145 (3): 250-257.

---. *L'exposition au plomb des nouveau-nés et les facteurs de risque associés : état de la situation dans la région de Québec* [Exposure to lead among newborns and the associated risk factors: progress report for the Québec region]. Québec: Ministère de la Santé et des Services sociaux, 1992.

---. 1993. Umbilical cord blood lead levels in the Québec City area. Arch Environ Health. 48 (6): 421-427.

Rhainds, M., et al. 1999. Lead, mercury and organochlorine compounds levels in cord blood in Québec, Canada. Arch Environ Health. 54: 40-47.

Saxena, D.K., et al. 1994. Blood and placental lead levels in an Indian city: a preliminary report. Arch Environ Health. 49: 106-110.

Tsuji, L.J.S., and E. Nieboer. 1997. Lead pellet ingestion in first nation Cree of the Western James Bay region of Northern Ontario, Canada: Implication for a nontoxic shot alternative. Ecosystem Health. 3: 54-61.

United States. Department of Labor. OSHA Regulation (Standards - 29CFR) Lead.-1910.1-25. 1999.

Watson, W.S., et al. 1986. Food iron and lead absorption in humans. Am J Clin Nutr. 44: 248-256.

Willers, S., et al. 1988. Relation between lead and cadmium in blood and the involuntary smoking of children. Scand J Work Environ Health. 14: 385-389.

Xiao-Ming, S., et al. 1997. Umbilical cord blood levels in Shanghai, China. Biomed Environ Sci. 10: 38-46.

Ziegler. E.E., et al. 1978. Absorption and retention of lead by infants. Pediatr Res. 12: 29-34.

APPENDIX 1: INTERNATIONAL COMPARISONS OF UMBILICAL-CORD BLOOD LEAD LEVELS

Author	Country	Year of Study	n	GM[4] (µmol/L)	Range (µmol/L)
Gershanik (1974)	USA	1972	218	0.453	--
Zetterlund (1977)	Sweden	1973-74	541	0.367	0.096-1.206
Winneke (1985)	FGR	1975-76	114	0.396	0.193-1.448
Tshuchiya (1984)	Japan	1974-78	95	0.405	0.050-2.504
Lauwerys (1978)	Belgium	1975-76	503	0.405	0.130-1.317
Rabinowitz (1982)	USA (Boston)	1979-81	11 837	0.318	0.000-1.785
McMichael (1986)	Australia	1979-82	500	0.487	0.469-0.506
Zarembski (1983)	England	1980-81	1 209	0.196	0.072-1.023
Ernhart (1985)	USA (Cleveland)	1981-82	178	0.275	0.125-0.709
Satin (1991)	USA (California)	1984	723	0.236	0.024-0.724
Declercq (1988)	France (Lille)	1986	144	0.230	0.000-1.340
Shucard (1988)	USA (Buffalo)	1987-88	802	0.183	0.072-0.965
Koren (1990)	Canada (Toronto)	1989	95	0.060	0.010-0.320
Our study	Canada (Québec)	1990	430	0.094	0.010-1.000

Source: Rhainds, M. and P. Levallois. 1993. Umbilical Cord Blood Lead Levels in the Québec City Area. Arch Environ Health. Vol. 48 (No. 6).

[4] Geometric mean

APPENDIX 2: QUESTIONNAIRE FOR INVESTIGATION OF LEAD SOURCES

(to be developed)

QUESTIONNAIRE: IDENTIFICATION OF ACTIVITIES AND ENVIRONMENTAL SOURCES LIKELY TO COUNTAIN LEAD

I. GENERAL INFORMATION

1. Name: _____

2. Address: _____

_____

3. Date of birth: _____/_____/_____
day month year

4. Year in which house was built: _____

5. Have other residents of this house already had lead content confirmed in their blood?

yes ☐ no ☐

Name	Age	Results	Date
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____
_____	_____	_____	_____

II. LIFESTYLE AND ACTIVITIES OF CHILDREN UNDER 15 YEARS (if the person with elevated blood-lead is older than 15 years, go to Section III.)

6. List the places where the child plays most frequently outside the home (around the house, in a shed, close to the dump, etc.):

_____

7. Does the child wash his or her hands before meals and snacks?

always ☐ often ☐ rarely ☐ never ☐

8. Does the child have a habit of:

placing his or her fingers or other objects into his or her mouth?

yes ☐ no ☐ If yes, specify: _____

chewing on objects such as:

toys	yes ☐	no ☐
furniture	yes ☐	no ☐
bedposts	yes ☐	no ☐
molding	yes ☐	no ☐
tin-lead solder	yes ☐	no ☐
rifle cartridges	yes ☐	no ☐
shotgun pellets	yes ☐	no ☐
placing the following in his or her mouth:		
paint flakes	yes ☐	no ☐
grass	yes ☐	no ☐
soil	yes ☐	no ☐

III. GENERAL ENVIRONMENT

9. Does the person with elevated blood lead consume tap water?

yes ☐ no ☐ If yes, daily quantity: _____ (litres)

10. What is the **principal** source of drinking water for the person with elevated blood lead?

water drawn directly from lake or river	☐	
house reservoir	☐	
untreated water (drawn upstream from treatment plant)	☐	
other	☐	Specify: _____

11. To your knowledge, does the plumbing in the home of the person with elevated blood lead contain pipes with lead solder?

yes ☐ no ☐

12. Over the past two years, has work been carried out for general renovation or for plumbing?

yes ☐ no ☐

If yes, specify: _____

13. Is there a resident of the house in which the person with elevated blood lead lives who normally performs the following in or near the house or in the shed?

metal foundry

yes ☐ no ☐ If yes, specify place: _____

recycling (breaking open) and/or storage of old batteries

yes ☐ no ☐ If yes, specify place: _____

radiator repair

yes ☐ no ☐ If yes, specify place: _____

recovery and storage of scrap metal

yes ☐ no ☐ If yes, specify place: _____

welding or soldering with lead alloys (e.g., tin-lead solder)

yes ☐ no ☐ If yes, specify place: _____

vehicle repair

yes ☐ no ☐ If yes, specify: _____

manufacture of ammunition, fishing sinkers, metallic objects or toys

yes ☐ no ☐ If yes, specify: _____

other ☐ Specify: _____

14. Does the person with elevated blood lead carry out any of the following activities in his or her home, in a workshop, at school or at work?

metal foundry

yes ☐ no ☐ If yes, specify place: _____

recycling (breaking open) and/or storage of old batteries

yes ☐ no ☐ If yes, specify place: _____

radiator repair

yes ☐ no ☐ If yes, specify place: _____

recovery and storage of scrap metal

yes ☐ no ☐ If yes, specify place: _____

welding or soldering with lead alloys (e.g., tin-lead solder)

yes ☐ no ☐ If yes, specify place: _____

vehicle repair

yes ☐ no ☐ If yes, specify: _____

manufacture of ammunition, fishing sinkers, metallic objects or toys

yes ☐ no ☐ If yes, specify: _____

work at or trips to the dump

yes ☐ no ☐ If yes, specify: _____

other ☐ Specify: _____

15. During the months before the first confirmation of elevated blood lead, did someone perform work involving sanding, scraping or stripping paint in the home or on objects such as bicycles, metallic toys, old furniture or boats?

yes ☐ no ☐ If yes, specify: _____

16. Did the person with elevated blood lead shoot with a rifle or shotgun during the months before the first confirmation of elevated blood lead?

yes ☐ no ☐ If yes, specify: _____

17. Does the person with elevated blood lead play with metallic objects such as fishing sinkers, empty rifle cartridges and shotgun pellets?

yes ☐ no ☐ If yes, specify: _____

18. Does the person with elevated blood lead consume meat from game killed with ammunition containing lead pellets?

yes ☐ no ☐ If yes, what type of game: _____

Are the pellets removed from the carcass before the meat is prepared or cooked?

yes ☐ no ☐

How is this meat normally prepared before it is consumed?

raw ☐ boiled ☐ dried ☐ roasted ☐

How many meals with this type of game does the person with elevated blood lead consume per year? _____ (approximate number)

19. How many persons smoke **inside** the home where the person with elevated blood lead lives? _____ (approximate number)

20. Does the person with elevated blood lead smoke?

yes ☐ no ☐ If yes, how many cigarettes per day: _____